American Journal of Industrial Medici. 19:303–316 (1991)

Asbestos-Related Disease in Public School Custodians

L. Christine Oliver, MD, Nancy L. Sprince, MD, and Reginald Greene, MD

A cross-sectional prevalence study of 120 public school custodians was carried out. The purposes were 1) to investigate the prevalence of asbestos-related disease in a group of custodians at risk for asbestos exposure in public schools and 2) to determine the proportion with disease attributable to exposures in school buildings. Medical and occupational histories, flow-volume loops, and posterior-anterior, lateral, and anterior oblique (AO) chest radiographs were obtained. Single breath DLCO was measured and chest auscultation performed. Mean age of subjects was 57 years and mean duration of work as a custodian, 27 years. Fifty-seven (47.5%) had no known or likely exposure to asbestos outside of their work as a school custodian (NOE). Pleural plaques (PP) occurred in 40 (33%) of the total group and 12 (21%) of the group with NOE. Pulmonary restriction (FVC <80% predicted, $FEV_1/FVC\% \geq 70$) occurred in 22 (18%) of the total group and 10 (17%) of those with NOE. DLCO was lower in the group with restriction. Multivariate analysis revealed significant associations ($p < 0.05$) between both PP and restriction and duration of asbestos exposure. AO radiographs increased PP detection by a factor of 1.9. Our results reveal PP prevalence in excess of background and pulmonary restriction in the study population, and indicate that PP are attributable to asbestos in schools. Findings with regard to pulmonary restriction need further investigation. Prudent management of asbestos in buildings is indicated for the prevention of related disease.

Key words: asbestos-containing material (ACM), building exposure, no outside exposure (NOE), pleural plaques, pulmonary restriction

INTRODUCTION

Health hazards of exposure to asbestos are well known [Becklake, 1976; Selikoff and Lee, 1978]. Cancer and other asbestos-related diseases have been reported in groups with heavy workplace exposures, in individuals with intermittent lower-level exposures such as those occurring in brake maintenance and repair, and in "bystanders" in areas where asbestos work was carried out [Becklake, 1976; Lorimer et al., 1970; Huncharek et al., 1989]. Household contacts of asbestos workers and

The Medical Services (Pulmonary and Critical Care Unit), Massachusetts General Hospital and Department of Medicine, Harvard Medical School, Boston, (L.C.O., N.L.S.).
Department of Radiology, Massachusetts General Hospital and Harvard Medical School, Boston, (R.G.).
Address reprint requests to L. Christine Oliver, MD, Pulmonary and Critical Care Unit, Massachusetts General Hospital, 55 Fruit Street, Boston, MA 02114.
Accepted for publication July 12, 1990.

residents living in proximity to asbestos sources have developed exposure-related disease [Epler et al., 1980; Newhouse and Thompson, 1965].

The risk posed by exposure to asbestos fibers in buildings has not been well studied. Friable asbestos-containing material (ACM) is found in public, residential, and commercial buildings. Its use in building construction began in 1890 in boiler insulation, and was expanded in 1910 to include pipe wrap and in 1935 to include sprayed- and trowelled-on insulation [Oppenheim-McMullen and Turner, 1986]. From the late 1940's to the early 1970's, ACM was used not only for insulation but also for decorative and acoustical purposes in buildings. Of the three fiber types (chrysotile, amosite, and crocidolite) in commercial use in the United States, chrysotile has accounted for over 90% of asbestos sold [National Research Council, 1984]. It is the predominant fiber type found in building ACM [Sawyer and Spooner, 1978]. Exposures occur as a result of 1) the release of fibers from friable ACM by general deterioration or attrition and by impact and 2) reentrainment by general activity and by such specific activities as sweeping or dusting [Sawyer and Spooner, 1978].

Potential fiber burden (dose) for an asbestos-exposed individual depends upon composition and condition of the ACM, proximity and relationship (active vs passive) to ACM, and duration of exposure. For building occupants such as office workers and public school teachers and students, dose is likely to be low—both absolutely and relative to custodians, who clean and maintain buildings. The latter group is exposed periodically to airborne concentrations of asbestos comparable to those that have caused disease in other occupational settings [Sawyer and Spooner, 1978]. Previous studies of health effects related to building exposures are limited, but asbestos-associated disease in excess of that found in the general population has been reported in building maintenance workers [Young et al., 1981; Cordier et al., 1987; Lilis et al., 1979]. Malignant pleural mesothelioma attributable to exposure to amosite in a building has been reported in a female office worker [Stein et al., 1989].

In 1983, a survey of ACM in Boston public school buildings was carried out in accordance with the regulatory requirements of the EPA [Federal Register, 1982; Survey for Friable Asbestos-Containing Materials in the City of Boston Public Schools, 1983]. A total of 129 schools was inspected and prioritized on the basis of presence or absence, type, location, and condition of ACM. Each room of each school was visited and the presence and condition of any sprayed-on fireproofing, pipecovering, or acoustical plaster possibly containing asbestos were recorded. Bulk samples of suspect materials were collected and analyzed by polarized light microscopy to confirm the presence of asbestos. Priority I schools had ACM in serious disrepair requiring prompt corrective action; Priority II, isolated damaged ACM; and Priority III, intact stable ACM. Survey results are shown in Figure 1. Subsequently, the United States Environmental Protection Agency (EPA) published the results of a national survey "to determine the extent of the use of friable asbestos-containing materials in buildings" [EPA, 1988]. Of the asbestos found, 70% was in pipe and boiler insulation, 14% in sprayed- or trowelled-on friable material, and 3% in ceiling tile. "Older" buildings were more likely to have asbestos-containing pipe and boiler insulation, while buildings constructed in the 1960's were more likely to contain sprayed- or trowelled-on ACM.

As these surveys indicate, the use of ACM in buildings in the United States has been widespread. Issues of risk have gone beyond occupants and custodial/mainte-

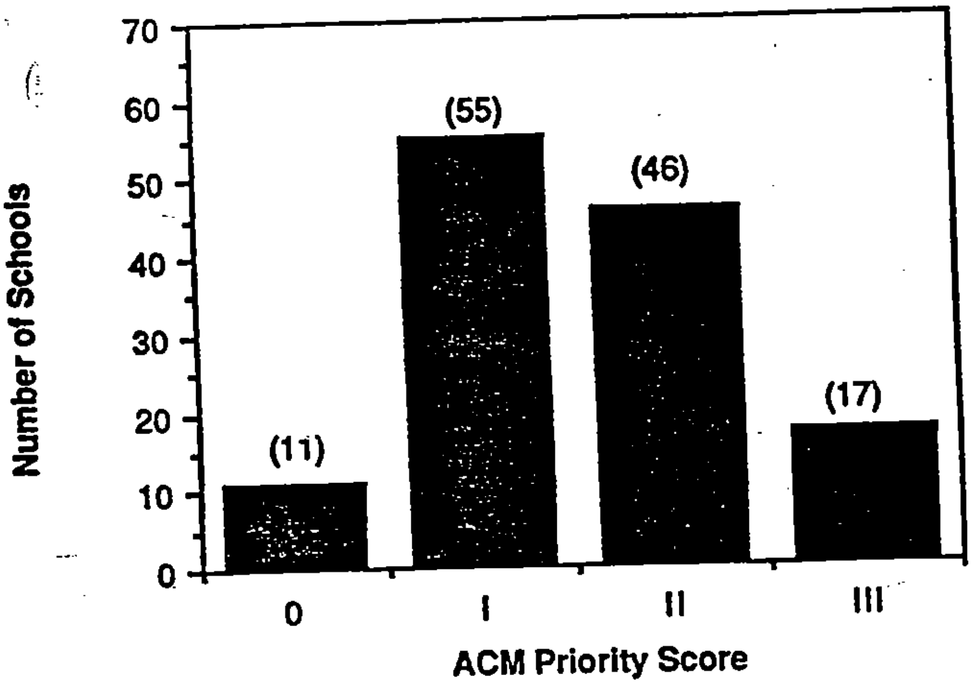

Fig. 1. Distribution of inspected schools by ACM Priority Score: 0 = no ACM, I = serious disrepair, II = isolated damaged, III = intact stable [Survey for Friable Asbestos-Containing Materials in the City of Boston Public Schools, 1983].

n… workers to affect building owners and developers, lenders, school departments, a… al, state, and federal governments. The purpose of the present study was to examine the occurrence of asbestos-related disease in custodians at risk for asbestos exposure in school buildings and to determine the proportion of disease attributable to building exposures.

METHODS

A cross-sectional morbidity study of 121 Boston public school custodians was carried out in 1987 (n=52) and 1988 (n=69). Subjects were active or retired members of the Boston Public School Custodians Association (BPSCA). In 1987, participation was limited to those with at least 20 years of service and in 1988, extended to those with at least 15 years of service.

Medical Evaluation

Questionnaire. Detailed occupational, smoking, and respiratory histories and information about current medication were obtained. The occupational history included questions about asbestos exposure: 1) at jobs held prior to the start of work as a Boston public school custodian; 2) at part-time jobs held during the period of work as a custodian; 3) as a result of household contact; and 4) as a result of geographic

proximity to an asbestos source, such as a shipyard or asbestos-manufacturing facility. Information was obtained about the time period and duration of employment at individual schools, and about frequency of performance of specific custodial tasks, such as sweeping and dusting of plaster and/or insulation dust, patching and/or removing torn insulation on pipes and/or boilers, and maintenance of boilers. Proximity to major boiler overhaul was recorded in terms of number and duration of occurrences. Latency was computed on the basis of date of hire for those custodians with no known exposure to asbestos outside of their work as a Boston public school custodian, and on the basis of date of first reported exposure for those with outside exposure. Where doubt existed, exposure was presumed and subjects were classified accordingly.

Smoking and respiratory histories were taken from the Epidemiology Standardization Projection questionnaire [Ferris, 1978]. Dyspnea was graded according to criteria set forth by the Medical Research Council of Great Britain [Fletcher et al., 1959].

Pulmonary function tests. Maximal expiratory flow-volume loops were obtained by using the Eagle One Spirometer with a 20k linear motion potentiometer attachment and microprocessor (Warren E. Collins, Braintree, MA). A minimum of three acceptable maneuvers was obtained. Maximal values for forced expiratory volume in 1 second (FEV_1), forced vital capacity (FVC), and FEV_1/FVC% were used. Prediction equations of Crapo and coworkers were used to derive predicted values [Crapo et al., 1981]. FEV_1 and FVC maneuvers were considered repeatable if the two best values for each agreed within 5% of the larger value or 100 ml [ATS, 1979]. A restrictive defect was defined as an FVC less than 80% of an age-, height-, and gender-based predicted value and an FEV_1/FVC% of 70 or greater, and an obstructive defect, as an FEV_1 less than 80% of the predicted value and an FEV_1/FVC% of less than 70.

The single-breath carbon monoxide diffusing capacity (DLCO) was measured by using the P.K. Morgan Modular Pulmonary Function Testing System (P.K. Morgan Co, Kent, Sussex, UK). The average value of two acceptable maneuvers was computed and used in data analysis [ATS, 1987]. Prediction equations of Miller were used to derive predicted values [Miller et al., 1983]. The single-breath DLCO maneuver was considered repeatable if the calculated difference between the two best values was less than 3 ml or 10% [ATS, 1987]. Hematocrit was obtained when single-breath DLCO % predicted was less than 80.

Chest radiographs. Posterior-anterior (PA), lateral, and right and left anterior oblique (AO) chest radiographs 41 × 43 cm were taken in an mobile X-ray unit at full inspiration, 110 KVp, and a standard distance of 183 cm. Interpretation was by a chest radiologist (RG) who is a NIOSH certified "B" reader, according to the ILO-1980 system of classification of radiographs [Classification of Radiographs of Pneumoconioses, 1981]. Each chest film was reread without knowledge of the initial reading by another "B" reader (LCO) or an experienced "A" reader (NLS). Differences were resolved by consensus reading. Definite pleural plaques were determined to be present when plaquelike thickening at the lung pleura interface along the lateral chest wall tangentially or along the en face rib margin was 2 mm or more, or when typical plaquelike thickening was present along either hemidiaphragm [Greene et al., 1984]. When no pleural plaques were detected or when minor or suspect pleural abnormalities were observed, the pleural findings were categorized as nor-

nal. The presence of pleural plaque was judged on the basis of four views of the c

. order to determine the extent to which AO radiographic views increased the ability of chest X-ray to detect pleural plaques above that of PA and lateral views alone, the two "B" readers reread 50 randomly selected PA/lateral film sets. The "reread" was carried out 1 to 2 years after the initial reading, without knowledge of the initial four-view reading. Classification based on two radiographic views was then compared to that based on four views.

Physical examination. Chest auscultation was performed by a physician (L.C.O. or N.L.S.). Dry end-inspiratory crackles that failed to clear with cough were recorded at each site at which they were heard. Height was measured without shoes; weight was self-reported.

Statistical Analysis

Student's *t* test was used to determine whether asbestos exposure, smoking, age, and body habitus differed significantly between those with and those without radiographic changes, pulmonary function test abnormalities, and dyspnea. Multivariate analyses were then used to examine the associations between exposure and pulmonary outcome, controlling for potential confounders. Logistic regression was used in the analysis of each dichotomous outcome and linear regression for each outcome described by a continuous measurement. A significance level of 0.05 was adopted.

RESULTS

ata analysis was carried out on 120 white males. One subject was excluded because of a history of medication with amiodarone, a drug associated with pulmonary fibrosis [Adams et al., 1988]. This individual had pleural plaques and interstitial fibrosis profusion 1/0 on chest roentgenogram. Of 197 eligible active school custodians, 102 (51.8%) were examined. Among those with at least 20 years since hire (n=113), participation rate was 70.8%. The number of retired custodians examined was 18. Information on the number eligible was not available.

Age and exposure characteristics of the study population are shown in Table I. Fifty-seven (47.5%) reported no exposure to asbestos outside of their usual work as a school custodian (NOE). Because of a high degree of interschool mobility, this group could not be classified more specifically with regard to level of asbestos exposure. Most "outside exposures" occurred as a result of work at shipbuilding and repair or in building construction. Compared to the group as a whole, those with NOE were younger, with shorter duration of asbestos exposure and latency. Distribution by smoking category and pack years of cigarette smoking was similar.

Chest Radiographs

Chest radiographs revealed pleural plaques (PP) in 40 (33.3%) subjects. In 39 cases, PP were bilateral; in one case, unilateral. Diffuse pleural thickening occurred unilaterally in one individual with a history of pleural effusion. This case was not

TABLE I. Age and Exposure Characteristics of Participants in Study of School Custodians, Boston, 1987–88

	Total	No outside exposure	p value[a]
n	120	57	
Age			
Mean	56.9 (7.8)[b]	55.2 (8.7)	0.0241
Range	38–77	38–77	
Years employed as a custodian			
Mean	26.1 (7.1)	25.7 (7.8)	0.5375
Range	6–46	6–46	
Latency (years)			
Mean	31.3 (8.3)	26.8 (7.7)	0.0001
Range	17–49	17–46	
Asbestos duration (years)			
Mean	28.6 (8.3)	25.7 (7.8)	0.0003
Range	6–48	6–46	
Pack years			
Mean	30.1 (31.5)	28.2 (29.0)	0.5337
Range	0–184	0–141	
Smoking category			
Nonsmoker	25 [20.8]	11 [19.3]	0.514
Exsmoker	63 [52.5]	33 [57.9]	$\chi^2 = 1.331$
Current smoker	32 [26.7]	13 [22.8]	

[a]No outside exposure vs. outside exposure.
[b]Standard deviations are in parentheses; percentages are in brackets.

included in the group with PP. Exposure associations are shown in Table II. Of the group with NOE, 12 (21%) had PP. The occurrence of PP was significantly higher in the group with a history of outside exposure to asbestos. Duration of asbestos exposure and latency were greater in those with PP compared to those without PP. Multivariate analysis revealed a significant association between the occurrence of PP and duration of asbestos exposure ($p = 0.0265$) and latency ($p = 0.0146$) for the group as a whole, controlling for smoking. Distribution of PP by years since first exposure is shown in Table III. For those with NOE, PP were associated with duration of work as a school custodian ($p = 0.0155$) and latency ($p = 0.0163$). Pack years of cigarette smoking were similar in the groups with and without PP ($p > 0.7$) in univariate analysis, and were not associated with PP ($p > 0.9$) in multivariate analysis.

Other factors were examined as potential confounders. Among those with PP, there were no reports of pneumonia, pleurisy, chest injury or surgery, or medication with agents known to be associated with pleural fibrosis. Subcostal fat may mimic PP, but weight (kg)/height (cm) ratios were similar in subjects with PP compared to those without PP—both for the group as a whole (0.49 vs. 0.48, $p = 0.4604$) and for the group with NOE (0.51 vs 0.48, $p = 0.1447$). One individual with outside exposure to asbestos gave a history consistent with rheumatoid arthritis. Two subjects gave a history of talc exposure. Both reported asbestos exposure outside of their usual work as a school custodian.

Three subjects (2.5%) had small irregular opacities on chest radiograph of profusion 1/0 by the ILO-1980 system. All reported outside exposure to asbestos: two

TABLE II. Chest Radiograph Results: Exposure Associations in Study of School Custodians

	PP[a]	No. PP	p value
	40 [33][b]	80 [67]	
Outside exposure			
Yes	28 [44.4]	35 [55.6]	0.007
No	12 [21.0]	45 [78.9]	$\chi^2 = 7.368$
Years employed as custodian			
Total group	27.7 (7.4)[b]	25.4 (6.9)	0.0891
NOE[c]	30.9 (7.9)	24.3 (7.2)	0.0082
Asbestos duration (years)			
Total group	31.0 (7.4)	27.4 (8.5)	0.0221
NOE	30.9 (7.9)	24.3 (7.2)	0.0082
Latency (years)			
Total group	34.4 (8.6)	29.7 (9.8)	0.0118
NOE	31.9 (8.2)	25.5 (7.0)	0.0088

[a]PP = Pleural plaques.
[b]Percentages are in brackets; standard deviations are in parentheses.
[c]NOE = No outside exposure to asbestos.

TABLE III. Distribution of Pleural Plaques by Latency in Study of School Custodians, 1987–88

	Total group		No outside exposure	
Latency (years)	n	PP[a]	n	PP
10–19	16	2 (12.5)[b]	11	1 (9.1)
20–29	39	9 (23.1)	28	3 (10.7)
30–39	38	17 (44.7)	14	6 (42.9)
40–49	27	12 (44.4)	4	2 (50.0)

$\chi^2 = 8.695$, $p = 0.034$

[a]PP = Pleural plaques.
[b]Percentages are in parentheses.

in a naval shipyard (4 years and 3 months, respectively) and one as an automobile mechanic (8 years). Their average duration of work as a school custodian was 28.3 years. Two were smokers and one, an exsmoker. Two had reduced single-breath DLCO (64% and 76% predicted) and crackles. One also had pulmonary restriction (FVC, 71% predicted; FEV_1/FVC% 82).

Pulmonary Function Tests

Mean values for measured parameters of pulmonary function are shown in Table IV. The number of subjects unable to perform repeatable ventilatory tests according to criteria set forth by the American Thoracic Society (ATS) was 10 (8.3%): for FEV_1, 4 (3.3%); for FVC, 5 (4.2%); for both FEV_1 and FVC, 1 (0.8%) [ATS, 1979]. The subject with both FVC and FEV_1 failure also had a restrictive defect. In no other case was FVC or FEV_1 test failure associated with a restrictive or obstructive defect.

TABLE IV. Pulmonary Function Test Mean Values in Study of School Custodians

		Outside exposure		
	Total	No	Yes	p value[a]
FVC				
Liters	3.8 (0.8)[b]	3.9 (0.8)	3.7 (0.7)	0.0873
% Predicted	85 (13)	86.6 (13)	82.9 (13.9)	0.1328
FEV_1				
Liters	2.9 (0.7)	3.0 (0.7)	2.8 (0.7)	0.0354
% Predicted	82 (17)	85.4 (15.4)	79.8 (17.2)	0.0652
FEV_1/FVC, %	77 (8)	78.5 (7.3)	75.9 (9.1)	0.0934
DLCO				
ml/min/mmHg	27.2 (5.5)	28.3 (4.8)	26.1 (6.0)	0.0278
% Predicted	87 (17)	90.1 (16.4)	84.2 (18)	0.0622
Restriction	22 [18.3]	10 [17.5]	12 [19]	0.832 $\chi^2=0.045$
Obstruction	19 [15.8]	8 [14]	11 [17.5]	0.608 $\chi^2=0.26$

[a] No outside exposure vs. outside exposure.
[b] Standard deviation are in parentheses; percentages are in brackets.

The group with a history of outside asbestos exposure had a trend toward lower FEV_1 and single-breath DLCO compared to those with NOE. The proportion with restriction and obstruction was not significantly different. In logistic regression analysis, restriction was positively associated with duration of asbestos exposure, both for the total group ($p=0.0044$) and for the group with NOE ($p=0.0420$), controlling for cigarette smoking. The single-breath DLCO, % predicted, was lower in the group with restriction (78.5 ± 21 vs. 88.9 ± 16, $p=0.0108$). Linear regression analysis revealed a negative association between DLCO, % predicted, and restriction ($p<0.02$), taking into account asbestos exposure and smoking. In the same model, DLCO (% predicted) was negatively associated with pack years for the total group ($p=0.0097$) but not for those with NOE ($p=0.4494$). The association with duration of asbestos exposure was not significant for either group ($p>0.7$). Low single-breath DLCO (< 80% predicted) was associated with restriction ($p=0.0323$), taking into account smoking and asbestos exposure. Associations with the latter two variables were not significant ($p=0.0798$ and 0.7089, respectively). Fourteen (11.7%) subjects were unable to perform repeatable single-breath DLCO maneuvers according to ATS criteria [ATS, 1987]. Eight reported NOE. Five (35.7%) had a restrictive ventilatory defect, compared to 17 (16%) of those who performed repeatable maneuvers. The percentage with an obstructive defect was similar in repeatable and non-repeatable subgroups (16% vs. 14.3%, respectively). Eight reported NOE.

Other

Twelve subjects (10.1%) reported symptoms of dyspnea Grade 2 or greater. The percentage was higher in the group with outside exposure (12.9%) compared to those with NOE (7%), but the difference was not significant ($p=0.287$, $\chi^2 = 1.135$). In multivariate analysis, dyspnea was associated with duration of asbestos exposure ($p=0.0346$), taking into account pack years of cigarette smoking ($p=0.7861$) and

eural plaques ($p=0.3651$). Crackles at two sites or more on chest auscultation cur 'n 6.7% ($n=8$) of the total group: 11.3% of the group with outside expo- re, of the group with NOE ($p=0.038$, $\chi^2 = 4.307$). Restriction occurred in ree subjects with crackles (37.5%) compared to 19 without crackles (17%) $=0.151$, $\chi^2 = 2.057$). There was no significant association between crackles and bestos exposure ($p=0.6647$), smoking ($p=0.5327$), or restriction ($p=0.2556$) in gistic regression analysis, controlling for each of the other variables.

One study participant subsequently developed malignant mesothelioma. In ad- tion to his work as a school custodian for 32 years, he reported working as a eetmetal worker in a naval shipyard for 6 months in 1942. Chest radiograph re- ealed PP.

ISCUSSION

Pleural plaques on chest radiograph are a marker of asbestos exposure, both ccupational and environmental [Albelda et al., 1982; Hillerdal, 1980]. Reported ackground prevalence figures vary from 0.003% in Australian adults over the age of), to 1.6% in nonexposed university employees in Paris, to 1.8% in a group of niversity laboratory technicians, maintenance personnel, and grounds workers in the ortheastern United States [Cordier et al., 1987; Gibbs, 1979; Epler et al., 1982]. The gnificance of the finding of PP on chest X-ray is uncertain. Associations with ecrement in lung function, with immunologic imbalance, and with increased risk for tal mortality, lung cancer mortality, and subsequent development of fibrosis of the ng parenchyma have been observed [Baker et al., 1985; Picado et al., 1987; Oliver : al., 1988; Sprince et al., submitted, 1990; Liddell and McDonald, 1980; Fletcher, 972; Finkelstein and Vingilis, 1984; McMillan and Rossiter, 1982].

'- the present study, asbestos-related PP on chest radiograph occurred in 33% .) of public school custodians who worked in buildings with friable ACM. hir. _ rcent ($n=12$) of those with PP had no known exposure to asbestos outside f their usual work as a school custodian. In that group, multivariate analysis revealed positive association between PP and duration of work as a custodian. None had mall irregular opacities on chest X-ray, and the number with dyspnea or crackles was mall. Pulmonary restriction occurred in 10 (17.5%).

The health risk associated with exposure to asbestos from friable ACM in uildings is undetermined. Levels of exposure depend upon: 1) the nature of the ssociation with ACM (direct vs. indirect); 2) the nature of the ACM (friable vs. onfriable); 3) the condition of the ACM (stable vs. damaged, deteriorating); and 4) ccessibility. Dose is a function of both level and duration of exposure. In general, ustodians in buildings with ACM are likely to accrue higher doses of asbestos than uilding occupants—such as teachers and students. For example, dusting in a uni- ersity library with a deteriorating sprayed asbestos ceiling produced an average level f 4.0 f/cm^3 for custodians, compared to 0.3 f/cm^3 for proximate library users Sawyer and Spooner, 1978]. Under quiet conditions in a university building with xposed friable ceilings containing 20% chrysotile asbestos, fallout resulted in levels f 0.02 f/cm^3, whereas cleaning and moving books in the stack area resulted in levels f 15.5 f/cm^3; removing a ceiling section, in levels of 12.7 f/cm^3; and sweeping and ry dusting, in levels of 1.6 f/cm^3 [Sawyer and Spooner, 1978]. These levels are far n excess of the current OSHA standard of 0.2 f/cm^3 and of ambient levels measured

by the EPA of 0.00007 f/cm^3 (2.3 ng/m^3) [Occupational Exposure to Asbestos, Tremolite, Anthophyllite, and Actinolite, 1986; EPA, 1988]. Our finding of radiographic evidence of asbestos-related pleural plaques in school custodians without other known exposure is consistent with these levels of asbestos exposure and with their mean duration of school employment of 26 years.

Other investigators have reported the occurrence of asbestos-related PP on chest radiograph in maintenance workers at risk for building exposures. Cordier and colleagues observed a higher prevalence of diaphragmatic PP and costophrenic angle blunting on chest x-ray in a group of maintenance workers with occupational exposure to asbestos in university buildings, compared to groups with environmental exposure in the same buildings and to those with no known exposure [Cordier et al., 1987]. "Maintenance" workers included skilled craftspeople (e.g., electricians, plumbers) and custodians. Difference in outcome between these two subgroups was not addressed, nor was mean duration of maintenance work given. The occurrence of PP on PA view of the chest was low (2.7%), in keeping with reported background levels of exposure—suggesting short duration and/or latency for the group overall. There was no excess of asbestos-related disease in the group with environmental exposure compared to the unexposed group. Small irregular opacities, profusion ≥1/0, occurred on chest x-ray in 20% of the latter group, however. Unknown asbestos exposure in that group would have the effect of reducing differences between groups and biasing results towards the null.

An excess of PP compared to background was observed by Young and associates in maintenance workers in nonasbestos factory buildings with ACM around steam and hot water pipes and boilers (6.1% [n = 13] compared to 0.003%) [Young et al., 1981]. Only one of the 13 with PP had prior asbestos exposure; 12 had worked at least 20 years. Lilis and colleagues observed asbestos-related PP on chest radiograph in 37.5% of maintenance workers in a large chemical plant in New Jersey [Lilis et al., 1979]. Although the group studied included skilled tradespeople, 58% (n = 108) reported "bystander" exposure only. Thirty percent of this group had abnormal chest radiographs. There was no significant difference in findings in those with and without a history of prior asbestos exposure.

The occurrence of PP in our study was comparable to that of Lilis et al. [1987] and in excess of that reported by Cordier and Young [Cordier et al., 1987; Young et al., 1981; Lilis et al., 1979]. Our prevalence was based on four radiographic views of the chest, while those by Cordier and Lilis were based on PA view alone. Young obtained both PA and lateral views. The extent to which anterior oblique (AO) chest radiographs aid in the diagnosis of asbestos-induced pleural disease varies, depending upon the diagnostic criteria applied and the underlying risk of the population examined [Classification of Radiographs, 1981; Baker and Greene, 1982]. In a study of asbestos-exposed construction workers, the addition of AO views increased the prevalence of PP 1.4 times, the effect being greater in those with mild disease [Baker and Greene, 1982]. In contrast, Sherman and associates reported minimal effect of AO views on the detection of PP in a group of pipefitters, with the prevalence of PP increasing 3.8% with the addition of AO radiographs [Sherman et al., 1988]. In that study, apparently the PA film was read initially and then the PA and AO films were read as a set at the same sitting. Thus, knowledge of findings on AO radiographs may have influenced interpretation of the PA film, resulting in augmentation of PP diagnosis on PA radiograph and reduction in apparent contribution of AO views. In the

resent study, the addition of AO radiographs increased PP prevalence by a factor of 1.9 ... 50 film sets. When four views were considered, 19 had PP compared to 10 wi... , and lateral views only were read. Thus, prevalence of PP was 38% using fou... views and 20% using two views in the subset that was reexamined. Two films interpreted as showing PP on the basis of PA and lateral views were considered normal on the basis of four views.

In 18% of our study population, pulmonary function tests revealed a restrictive defect. Occurrence was similar in those with and without outside exposure to asbestos and was associated with duration of asbestos exposure in both groups. This prevalence of restriction was greater than that observed by Lilis and associates, who found restriction (FVC % predicted ≤ 79) in 5.4% of maintenance workers examined [Lilis et al., 1979]. Cordier and colleagues found FVC and FEV_1 to be significantly lower in the group with occupational exposure to asbestos in buildings, but neither they nor Young reported on the occurrence of restriction in their study populations [Young et al., 1981; Cordier et al., 1987]. The significance of our finding is unclear. Single-breath DLCO was lower in the group with restriction, and was negatively associated with restriction in multivariate analysis. Restriction occurred more commonly in those with crackles, but the number with both abnormalities was small. These findings suggest the presence of occult interstitial fibrosis, possibly related to exposure to asbestos and/or some other as yet unidentified substance in the work environment of school custodians.

Potential weaknesses of the present study are: 1) the voluntary nature of participation; 2) lack of an unexposed comparison group; and 3) possible misclassification with regard to outside exposure status. Voluntary participation can introduce selection bias. Custodians who volunteered to participate may have been those at greatest risk for development of disease because of greater duration and intensity of asbestos exposure and longer latency. To address this issue, we characterized non-p... ants by age and seniority at the time of the study. Participants and non-pa... pants were similar in age: 55.4 ± 7.2 years vs. 54.5 ± 9.4 years, $p=0.4648$. Years since hire (seniority) were 19.5 ± 5.8 for non-participants, while years worked as a custodian were 25.4 ± 6.8 for participants ($p=0.0001$). This difference was largely attributable to the difference in the number whose eligibility was determined on the basis of 15 years seniority. For participants, the number was 22 (18.3%); for non-participants, 61 (64.9%). For those with at least 20 years seniority, participation rate was higher and participants resembled non-participants with regard to both age (56 and 59 years, respectively; $p = 0.0587$) and years of custodial work (28 and 26 years, respectively; $p = 0.1536$). The occurrence of PP was not significantly different in participants with at least 20 years of service (31.2%) compared to those with less (22.7%) [$\chi^2 = 0.604$, $p = 0.437$]. The prevalence of restriction was also similar in these two groups: 15% vs. 13.6%, respectively; $\chi^2 = 0.026$, $p = 0.873$. Thus, although participants had worked more years as custodians, it is unlikely that this difference significantly affected the results.

An external referent group was not available for comparison. On the basis of: 1) historical information obtained about time period and duration of employment by school and about frequency of performance of specific custodial tasks; and 2) The priority Score assigned to each school, an asbestos exposure index was constructed for each subject in an attempt to identify an internal referent group with little or no asbestos exposure. A high degree of interschool mobility and homogeneity of the

study population with regard to performance of custodial tasks made the identification of such a comparison group impossible. Fortunately, data were available on prevalence of PP in a group of 717 laboratory, maintenance, and grounds personnel in a large university in Boston [Epler et al., 1982]. None had known exposure to asbestos at the university. Prevalence of PP was 1.8% overall, and 0.7% if eight subjects with prior occupational asbestos exposure were excluded. These figures are in keeping with other reported figures of background plaque prevalence [Young et al., 1981; Cordier et al., 1987]. Using this group for comparison, we conclude that the occurrence of PP in our study population was excessive, taking into account the increment attributable to AO radiographs.

In order to minimize misclassification with regard to outside asbestos exposure, we contacted those subjects who reported "Don't know" in response to specific questions about asbestos exposure or who reported work at jobs for which the likelihood of asbestos exposure was uncertain. In these cases, telephone interviews were conducted by one of the authors (L.C.O.) to obtain more detailed information about job responsibilities and workplace exposures. When doubt about asbestos exposure status could not be resolved, subjects were considered to have had outside exposure for purposes of data analysis.

CONCLUSIONS

The present study reveals PP on chest radiograph and spirometric evidence of pulmonary restriction in a group of public school custodians. PP prevalence is in excess of background, taking into account the incremental value of AO radiographs. Results indicate that PP are attributable to asbestos exposure in schools and suggest the presence of occult interstitial fibrosis of the lung associated with work as a custodian. Our findings point to the need for improved medical surveillance of this population of workers. Prudent management or removal of friable ACM in buildings is important to the prevention of asbestos-related disease.

ACKNOWLEDGMENTS

We gratefully acknowledge the support of the Boston Public School Custodians Association and the Boston School Department. We further acknowledge the technical assistance of Barry Callahan, Paul Pappagianopoulos, and Henry Clausen, the clerical and administrative assistance of Beverly Snell, and the work of our computer programmer Mary Hyde.

REFERENCES

Adams GD, Kehoe R, Lesch M, Glassroth J (1988): Amiodarone-induced pneumonitis. Assessment of risk factors and possible risk reduction. Chest 93(2):254–262.

Albelda SM, Epstein DM, Gefter WB, Miller WT (1982): Pleural thickening: Its significance and relationship to asbestos dust exposure. Am Rev Respir Dis 126:621–624.

ATS statement (1979): Snowbird workshop on standardization of spirometry. Am Rev Respir Dis 119: 831–838.

ATS statement (1987): Single breath carbon monoxide diffusing capacity (Transfer factor). Am Rev Respir Dis 136:1299–1307.

er EL, Dagg T, Greene RE (1985): Respiratory illness in the construction trades. The significance of asbestos-associated pleural disease among sheet metal workers. J Occup Med 27:483–489.

er F' Greene R (1982): Incremental value of oblique chest radiographs in the diagnosis of asbestos- d pleural disease. Am J Ind Med 3:17–22.

H . (1976): Asbestos-related diseases of the lung and other organs: Their epidemiology and implications for clinical practice. Am Rev Respir Dis 114:55–95.

sification of Radiographs of Pneumoconioses (1981): Med Radiogr Photogr 57:1–17.

dier S, Lazar P, Brochard P, Bignon J, Ameille J, Proteau J (1987): Epidemiologic investigation of respiratory effects related to environmental exposure to asbestos inside insulated buildings. Arch Environ Health 42:303–309.

po RO, Morris AH, Gardner RM (1981): Reference spirometric values using techniques and equipment that meet ATS recommendations. Am Rev Respir Dis 123:659–664.

PA Study of Asbestos-Containing Materials in Public Buildings. A Report to Congress" (1988): U.S. Environmental Protection Agency. Washington, D.C.

er GR, Fitzgerald MX, Gaensler EA, Carrington CB (1980) Asbestos-related disease from household exposure. Respiration 39:229–240.

er GR, McLoud TC, Gaensler EA (1982): Prevalence and incidence of benign asbestos pleural effusion in a working population. JAMA 247:617–622.

leral Register. 47 RF23360–23389, May 27, 1982.

ris BG (1978): Epidemiology standardization project. Am Rev Respir Dis 118:1–87.

kelstein MM, Vingilis JJ (1984): Radiographic abnormalities among asbestos-cement workers. An exposure-response study. Am Rev Respir Dis 129:17–22.

tcher CM, Elmes PC, Fairbairn AS, Wood CH (1959): The significance of respiratory symptoms and the diagnosis of chronic bronchitis in a working population. Br Med J 2:257–266

tcher DE (1972): A mortality study of shipyard workers with pleural plaques. Br J Ind Med 29: 142–145.

bs GW (1979): Etiology of pleural calcification: A study of Quebec chrysotile asbestos miners and millers. Arch Environ Health 34:76–83.

ns LC (1990): Asbestos exposure and asbestos-related pleura and parenchymal disease: associations with immune imbalance. Submitted for publication.

ene R, Boggis C, Jantsch H (1984): Asbestos-related pleural thickening: Effect of threshold criteria on interpretation. Radiology 152:569–573.

lerdal G (1980): Pleural plaques. Occurrence, exposure to asbestos, and clinical importance. Acta rsitatis Upsaliensis 363:158–178.

m I, Muscat J, Capotorto JV (1989): Pleural mesothelioma in a brake mechanic. Br J Ind Med 9–71.

ldell FDK, McDonald JC (1980): Radiological findings as predictors of mortality in Quebec asbestos workers. Br J Ind Med 37:257–267.

is R, Daum S, Anderson H, Sinota M, Andrews G, Selikoff IJ (1987): Asbestos disease in maintenance workers of the chemical industry. Ann NY Acad Sci 330:127–135.

rimer WV, Rohl AN, Miller A, Nicholson WJ, Selikoff IJ (1970): Asbestos exposure of brake repair workers in the United States. Mount Sinai J Med 38:492–500.

cMillan GHG, Rossiter CE (1982): Development of radiological and clinical evidence of parenchymal fibrosis in men with non-malignant asbestos-related pleural lesions. Br J Ind Med 39:54–59.

iller A, Thornton JC, Warshaw R, Anderson H, Teirstein AS, Selikoff IJ (1983): Single breath diffusing capacity in a representative sample of the population of Michigan, a large industrial state. Am Rev Respir Dis 127:270–277.

tional Research Council/National Academy of Sciences (1984): "Asbestiform Fibers—Nonoccupational Health Risks" Washington, D.C. National Academy Press.

whouse ML, Thompson H (1965): Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. Br J Ind Med 22:261–269.

Occupational Exposure to Asbestos, Tremolite, Anthophyllite, and Actinolite" (1986): US Department of Labor/Occupational Safety and Health Administration. Federal Register 29 CFR Parts 1910 and 1926. 5(119).

liver LC, Eisen EA, Greene R, Sprince NL (1988): Asbestos-related pleural plaques and lung function. Am J Ind Med 14:649–656.

ppenheim-McMullen J, Turner B (1986): Facing the asbestos menace. Progressive Builder. In Spengler

JD, Ozkaynak H, McCarthy JF, Lee H (eds) (1989): "Summary of Symposium on Health Aspects of Exposure to Asbestos in Buildings Proceedings. Symposium on Health Aspects of Exposure to Asbestos in Buildings." Cambridge: Harvard University Energy and Environmental Policy Center.
Picado C, Laporta D, Grassino A, Cosio M, Thibodeau M, Becklake MR (1987): Mechanisms affecting exercise performance in subjects with asbestos-related pleural fibrosis. Lung 165:45–57.
Sawyer RN, Spooner CM (1978): "Sprayed Asbestos-Containing Materials in Buildings. A Guidance Document." U.S. Environmental Protection Agency. EPA 450/2-78-014.
Selikoff IJ, Lee DNK (1978): "Asbestos and Disease." New York: Academic Press.
Sherman CB, Barnhart S, Rosenstock L (1988): Use of oblique chest roentgenograms in detecting pleural disease in asbestos-exposed workers. J Occup Med 30:681–683.
Sprince NL, Oliver LC, Mcloud TC, Eisen EA, Christiani DC, Ginns LC (1990): Asbestos exposure and asbestos-related pleural and parenchymal disease: Associations with immune imbalance. Submitted, Am Rev Respir Dis.
Stein RC, Kitajewska JY, Kirkham JB, Tait N, Shinha G, Rudd RM (1989): Pleural mesothelioma resulting from exposure to amosite asbestos in a building. Respir Med 83:237–239.
"Survey for Friable Asbestos-Containing Materials in the City of Boston Public Schools" (1983): Hygeia Inc., Boston MA.
Young I, West S, Jackson J, Cantrell P (1981): Prevalence of asbestos-related lung disease among employees in non-asbestos industries. Med J Aust 1:464–467.